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Omaha Childhood Blood Lead and Environmental Lead: A Linear Total Exposure Model

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The majority of experimental and population studies of blood lead (PbB) and environmental lead, including the Omaha study, have utilized the Goldsmith–Hexter log–log or power function model. Comparison was made of the log–log model and a linear model of total exposure to describe the Omaha Study of 1074 PbBs from children ages 1–18 years as related to air (PbA), soil (PbS), and housedust (PbHD) lead. The data fit of the linear model was statistically equivalent to the power model and the predicted curves were biologically more plausible. The linear model avoids the mathematical limitations of the power model which predicts PbB zero at PbA zero. From the Omaha data, this model, $\ln PbB = \ln (\beta_0 + B_1 PbA + B_2 PbS + \beta_3 PbHD)$ predicts that PbB increases 1.92 μg/dl as PbA increases 1.0 μg/m^3. Since PbS and PbHD increase with PbA, however, the increases in total exposure predict a PbB increase of 4–5 μg/dl as PbA increases 1.0 μg/m^3.

INTRODUCTION

Determination of the acceptable level of air-borne lead requires critical evaluation of the contribution of air lead to total environmental exposure. Air lead does not remain indefinitely suspended and must ultimately correlate with the lead content of food, water, soil, house dust, hand dust, and other forms of ingested lead, the main source of the lead burden in children. Experimental exposure studies that relate blood lead to air lead as delivered in a chamber or by direct inhalation may underestimate the dose response to environmental changes in air lead. Population studies that relate blood lead to air lead under varying conditions of lead from current or previous atmospheric fall out and other sources

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may yield estimates of dose response that are biologically more relevant to the study population.

There are relatively few large scale population studies of the blood lead of children as related to both atmospheric lead and the surface lead of soil and dust, and only a limited number of models have been used to describe the data. A dose–response model that predicts the change in the blood lead of a population to increases or decreases in air lead is a prerequisite to the determination on air quality standard for lead. This is a reanalysis of the Omaha Study (Angle and McIntire, 1979) to examine a linear model of the total exposure for the relation of childhood blood lead to air, soil, and house dust lead.

The majority of clinical investigations (Azar *et al.*, 1975; Griffin *et al.*, 1975; Rabinowitz *et al.*, 1977) and of population studies (Johnson *et al.*, 1976; Roberts *et al.*, 1974; Roels *et al.*, 1980; Snee, 1982b; Tepper and Levin, 1975; Yankel *et al.*, 1977), including our previous analyses (Angle and McIntire, 1979, 1982), have applied the power function model of Goldsmith and Hexter (1967). Since blood lead has a minimum value of zero, the distribution of blood lead in any population has a skew to the right; a logarithmic transformation of blood lead yields a close to normal distribution considered more appropriate for statistical analysis. A non-linear transformation of the environmental data reflects the less rapid response of blood lead to inhaled or ingested lead at the highest exposure levels (Hammond *et al.*, 1981). McCaughran (1975) pointed out that a major mathematical deficiency of the log–log model is that it predicts a blood lead of zero at an air lead of zero. Snee (1982a, 1982b) has thus proposed linear models of total exposure, and suggests that linear models also avoid an overestimation of the steep response of blood lead at air lead below 1.0 $\mu g/m^3$.

The Omaha Study is relatively unique in its size, population characteristics, and duration of sampling. It is also representative of large scale community studies that relate the wide range of all individual levels of blood lead to environmental samples collected at central sites and often averaged over time. The data consist of 1074 samples of blood lead from 831 urban and suburban children 1–18 years old, 1970–1977, with concurrent values for air, soil, and dust lead. During the time of the study, Omaha had three identified lead emission sources but only 153 samples (14%) were from children attending grade school adjacent to one of these sources, a small battery-making plant. To be consistent with other population studies, the earlier analysis examined the log–log model although a linear regression appeared to provide an equivalent prediction of blood lead. The broad range of the population and the typical environmental sampling techniques of the Omaha Study provide representative data for investigation of an alternate linear model to describe the blood lead response of a general population of children and youth to environmental lead.

METHODS

As previously described (Angle and McIntire, 1979, 1982), the study was conducted 1971–1977 in three areas of Omaha: urban commerical (C) in the vicinity of a small battery plant, urban mixed (M) in a residential area contiguous with downtown Omaha, and surburban (S). Children are categorized as ages 1–5 or preschool (n = 242 samples), and 6–18 years (n = 832 samples). The preschool

group was sampled only in February, April, and July–August of 1974, a time interval characterized by the typically high air leads of winter and low air leads of summer as well as a paradoxical suburban increase in air lead. Since there was an increase in the summertime blood leads of both urban and suburban children, the preschool data is difficult to interpret, although typical of the artifacts present in community studies. All 153 samples at Site C (battery plant) were from 6- 12-years olds. Almost all children at sites C and M were black and lived in designated poverty areas. Almost all suburban children were white. In 1976–77 third grade children were bused from the suburbs to the school at site C; since the PbB of these children remained consistently lower than that of local children in both November 1976 and May 1977, the children bused from the suburbs are classified as suburban.

Blood lead. In 1971 all samples were venous blood collected in Pb-free heparinized B-D vacutainers and analyzed by the macro method of Farrelly and Pybus by atomic absorption spectroscopy. In 1972 and 1973 all subjects had both a macro venous blood lead and a micro venous assay by the Delves cup technique. Analyses done at this time showed the micro assay to be consistently higher by 4.0 μg/dl and the macro assays of 1971 were corrected to the higher micro values. All assays from 1972 to 1977 were done on venous blood by the Delves micro cup technique using a Perkin Elmer 300 Atomic absorption spectrometer. All assays were done in duplicate employing the method of addition for each sample. Blood lead (PbB) as measured in 1232 samples from 831 subjects had a close-to-normal distribution after logarithmic transformation. The geometric mean (GM) was 21.47 μg/dl, geometric standard deviation (GSD) 1.395, skew 0.36, and kurtosis 3.0. At site C, the blood lead (GM ± GSD) of 24.8 ± 1.4 was significantly higher than the value of 22.0 ± 1.4 at site M and the 18.0 ± 1.3 at S. Blood leads decreased significantly from 1971 to 1975 but not from 1975 to 1977. Sampling analysis, the difference between repeat samples from one individual, accounted for 38% of the total variance. Concordant environmental data for air, soil, and house dust correlates were available for 1074 of the 1232 blood samples, eliminating 166 of the repeat blood samples obtained from the 831 subjects. All of the repeat blood samplings employed in the data analysis had a different air lead than prior samplings so that each of the 1074 samples represents a different data set.

Air lead. Air lead (PbA) samples were 24-hr collections at 6-day intervals in Hi-Vol samplers (0.1–10 μm particle size) at 15 ft elevation at schools in each of the three areas. Analysis was by atomic absorption. The seasonal patterns showed peak values in January through March, consistent with climatic inversions. The annual geometric mean PbA (μg/m^3) at each site was

	C	M	S
1970	1.66	1.44	0.73
1972	0.37	0.32	0.26
1973	0.46	0.32	0.22
1974	0.04	—	0.04
1976	0.13	0.16	—
1977	0.78	0.62	—

TABLE I
ENVIRONMENTAL AND BLOOD LEADS AT EACH SITE

Site	*n*	PbB[a] (μg/dl)	PbA[b] (μg/m³)	PbS[c] (μg/g)	PbHD[d] (μg/g)
		Ages 1–5 years			
C	0	—	—	—	—
M	204	25.6 ± 0.6	0.26 ± 0.01	213 ± 7	653 ± 13
		(10.8–69.8)	(0.02–0.40)	(118–353)	(190–860)
S	38	14.6 ± 0.5	0.37 ± 0.02	110 ± 2	567 ± 56
		(9.9–22.7)	(0.02–0.50)	(85–118)	(110–860)
		Ages 6–18 years			
C	153	26.3 ± 0.7	0.86 ± 0.04	519 ± 15	624 ± 12
		(12.5–63.0)	(0.30–1.70)	(35–815)	(186–873)
M	467	22.2 ± 0.3	0.63 ± 0.03	389 ± 15	264 ± 6
S		(8.2–62.9)	(0.02–1.50)	(87–870)	(160–550)
	212	19.3 ± 0.04	0.29 ± 0.01	97 ± 2	324 ± 6
		(10.2–44.5)	(0.02–0.50)	(40–385)	(99–681)

Note. All values given as arithmetic mean ± standard error of the mean with the range in parentheses.

[a] All 1074 blood leads (PbB) with complete environmental correlates were used; outliers were not excluded.

[b] Air lead (PbA) is the average of five 24-hr samples/month at a single Hi-Vol collector for each site, during the month that PbB obtained; if not available, PbA of the nearest of the 3 prior months was assigned.

[c] Soil lead (PbS) is the average of four 2-in. cores taken halfway between the building and lotline. For 37 children this is their own home; for all others it is the sampling at their school (one to five samplings at 20 schools) at the shortest interval from the PbB.

[d] Housedust lead (PbHD) is a single sample from the vacuum cleaner or a composite brushing of sills and floor. For 37 children this is their own home; all others were assigned the sampling at their school at the shortest interval from the PbB.

Each blood lead sample was assigned a PbA representing the mean for the month of collection or the closet sampling in the prior 3 months. The average monthly values assigned to each blood lead (Table 1) do not indicate the variability evidenced by the range of concentrations of individual samples of PbA μg/m³ at each site:

	N	Range
C	180	0.005–4.85
M	118	0.020–2.32
S	141	0.005–1.64

Dustfall lead. Dustfall lead (PbDF) was collected at each of the air collection sites, in plastic containers at 4 ft height. Analysis was by atomic absorption. The annual geometric means, expressed as milligrams per square meter per 30 days collection, had the same rank order as air lead

	C	M	S
1973	26	11	7
1974	17	6	3
1976	5	1	—
1977	34	8	—

Although not used for the final analysis because of the limited sampling, the highest range of dustfall lead ($mg/m^2/30$ days), was found at site C:

	N	Range
C	9	5.3–33.9
M	10	1.2–11.6
S	7	1.3– 9.4

Soil lead. Soil lead (PbS) was sampled as 2-in. cores taken halfway between the building and lot line on all four sides and reported as the arithmetic mean for each site. In 1972 soil and housedust were obtained at 37 homes and utilized for correlation with individual blood leads. All other blood leads were assigned an averaged soil lead from the sampling at each subject's school at the time closest to the blood sampling (Table 1). A total of 20 sites were sampled one to five times. The range of PbS, micrograms per gram, of individual cores, at each site was

Site	*N*	Range
C	69	56–1615
M	56	20–4792
S	51	16–341

The higher values at site M may represent individual variables in building construction or finish or may relate to prior fall out from the two major lead sources located 2 miles ESE of site M. The extreme values at site M are not reflected in the average values listed in Table 1.

House dust lead. House dust lead (PbHD) was obtained at the same sites as PbS. Almost all samples were obtained by emptying the vacuum cleaner bag; when this was not available, a dustpan was used to collect a composite sweeping of the floor and windowsills. For 37 samples the assigned values are those of child's own home; all other samples were the PbHD of the subject's school as obtained at the shortest interval from PbB (Table 1). These assigned values show less variability than the range of the individual samples of PbHD μg/g:

Site	*N*	Range
C	26	76–5571
M	14	76–860
S	26	18–845

Other sources. A household study of water and milk lead showed all bottled milk samples, which came from a common milk shed, to have less than 40 μg/liter. The water samples, tested on 30-day collections at 20 homes, had less than 10 μg/liter of lead, consistent with the pH 8.5–9.5 of Omaha tap water. Food analysis was not part of the study but all three grocery chains in Omaha are equally represented in the three areas.

Statistical analysis. The choice of a statistical analysis method depends on the assumptions one is willing to make about the nature of the unexplained variability in the data. We assume that the random variability in blood lead concentrations is more likely to be multiplicative than additive, i.e., for some model that predicts the geometric mean (GM), the individual PbB is

$$\mathrm{PbB} = (\mathrm{GM}) \exp(\epsilon), \tag{1}$$

where ϵ is the random error, here assumed to be normally distributed with mean 0 and unknown standard deviation σ. Thus

$$\ln \mathrm{PbB} = \ln \mathrm{GM} + \epsilon. \tag{2}$$

The regression model depends on the assumed form for GM. The Goldsmith–Hexter model or its generalizations assume that GM is a *power function* of lead concentrations in various media, including air lead (PbA), lead in soil (PbS), house dust (PhHD), etc., so

$$\mathrm{GM} = \exp(\beta_0)\,(\mathrm{PbA})^{\beta_1}\,(\mathrm{PbS})^{\beta_2}\,(\mathrm{PbHD})^{\beta_3} \tag{3}$$

or

$$\ln \mathrm{GM} = \beta_0 + \beta_1 \ln \mathrm{PbA} + \beta_2 \ln \mathrm{PbS} + \beta_3 \ln \mathrm{PbHD}. \tag{4}$$

The disadvantage of the power function model of Eq. (3) and its logarithmic form, Eq. (4) is that it predicts GM = 0 if *any* of the environmental sources is absent, with correspondingly unreasonable predictions for small exposures of any sort. Thus Snee (1982a) suggested as an alternative a *total exposure* model which in our notation is

$$\mathrm{GM} = (\beta_0 + \beta_1\,\mathrm{PbA} + \beta_2\,\mathrm{PbS} + \beta_3\,\mathrm{PbHD})^{\lambda} \tag{5}$$

for some constant λ. The *linear exposure* model sets $\lambda = 1$. However, this model cannot be fitted using a standard linear regression program since ln GM is now intrinsically nonlinear,

$$\ln \mathrm{GM} = \ln(\beta_0 + \beta_1\,\mathrm{PbA} + \beta_2\,\mathrm{PbS} + \beta_3\,\mathrm{PbHD}). \tag{6}$$

This linear model was fitted to our data using the least squares program NLIN in the Statistical Analysis System (Ray, 1982). A grid search among initial estimates of the β parameters was improved iteratively to obtain the estimates in Table 2. The power model Eqs. (3) and (4) were fitted to the multivariate regression model utilizing the natural logarithms.

TABLE 2
REGRESSION COEFFICIENTS FOR FITTED MODELS

	Age group			
	6–18 years (n = 832)		1–18 years (n = 1074)	
	Power[a]	Linear[b]	Power	Linear
β_0	1.9547 (0.1335)	16.2136 (0.4201)	1.5202 (0.1067)	15.6715 (0.0398)
β_1 (Air)	0.0222 (0.0099)	4.4001 (0.6092)	−0.0223 (0.0080)	1.9175 (0.6001)
β_2 (Soil)	0.1253 (0.0132)	0.00457 (0.00090)	0.1435 (0.0121)	0.0068 (0.0010)
β_3 (House dust)	0.0772 (0.0231)	0.00336 (0.00140)	0.0127 (0.0163)	0.0072 (0.0009)
Residual sum of squares (n units)	68.2177	64.6089	96.3599	96.5089
R^2	0.2204	0.2616	0.1995	0.1983

Note. Values in parentheses represent standard errors of the estimates of coefficients.
[a] $\ell n\ PbB = \beta_0 + \beta_1\ \ell n\ PbA + \beta_2\ \ell n\ PbS + \beta_3\ \ell n\ PbHD$
[b] $\ell n\ PbB = \ell n\ (\beta_0 + \beta_1\ PbA + \beta_2\ PbS + \beta_3\ PbHD)$

RESULTS AND DISCUSSION

Table 2 defines the minimal differences in data fit of the power function model and the linear model of total exposure. For ages 1–18 years, both models have 1074 observations and use four parameters (here labeled β_0–β_3, although the interpretations are quite different). The linear model has an equivalent fit as measured by a similar residual sum of squares (96.4 vs 96.5 in natural log units), and a comparable R^2 of .20. Direct comparison of these models by the usual large-sample F test or χ^2 likelihood ratio criterion (Gallant, 1975) is not appropriate since the models represent separate families of curves rather than a hierarchical family, and more complicated tests are needed (Pereira, 1978). An informal comparison shows that there is little difference in the ability of the models to fit the data.

The linear model, however, avoids the negative coefficient for air lead. The biologic implausibility of this coefficient is evident in the negative slope predicted by the power model for the change in PbB μg/dl in response to a change in PbA of 1.0 μ g/m^3 at a constant PbS and PbHD of 200 μg g. for ages 1–18 years:

PbA	PbB (power model)	PbB (linear model)
0.1	11.02	18.66
1.0	10.46	20.39
2.0	9.78	22.31

The power model predicts a decrease in PbB of 0.68 μg/dl as air lead increases from 1 to 2 μg/m^3; this is inconsistent with the overall distribution of the observations. The linear model predicts an increase in PbB of 1.92 as PbA increases

from 1 to 2 $\mu g/m^3$, a slope consistent with numerous clinical and community studies (Snee, 1982a, 1982b).

For ages 6–18 years, with 832 data sets, the linear model may provide a slightly better fit than the power model with a residual sum of the squares of 64.6 vs. 68.2 and R^2 of .26 vs .21. Although the two models provide an equivalent fit for the data, the linear model describes a considerably steeper blood:air slope for ages 6–18 years (4.40 vs. 0.33) at a constant level of PbS and PbHD of 200 $\mu g/g$:

PbA	PbB (power model)	PbB (linear model)
0.1	19.62	18.24
1.0	20.64	22.20
2.0	20.97	26.60

The variability in the estimated regression coefficients and in the predicted blood:air slopes is partly due to the fact that air lead, soil lead, and dust lead are highly correlated. Site S has the lowest geometric mean of each, Sites M and C have higher values (Table 1). This multicollinearity may lead to inflated estimates of standard error of the coefficients and to possible instability in the values (and even the signs) of the estimated coefficients. Lead input from water is negligible in Omaha, and lead from the diet is presumed to be similar at all sites, prior to local contamination. The major sources of collinearity are shown by the Pearson correlations of the environmental variables:

	PbS	PbHD	PbDF
PbA	0.37	0.17	0.20
PbS		0.27	0.35
PbHD			0.57

This multicollinearity partly explains why the inclusion of the dustfall assays, available for only a limited number of subjects, did not strengthen the predictive power of the total exposure model.

The linear total exposure model allows estimation of changes in the blood lead of Omaha children exposed to moderate changes in air lead with and without secondary changes in surface deposition. The immediate effect, projected as the response to the direct inhalation of air lead, is an increase in blood lead of 1.92 $\mu g/dl$ as air lead increases 1 $\mu g/m^3$. The higher soil and house dust lead in the urban areas of high air lead, however, supports an associated increase in ingested lead from the fallout of lead-bearing particulates. The many variables of age, sex, race, socio-economic status, housing construction, lead content of paint and plaster, surface deterioration, household cleanliness, and personal hygiene preclude accurate estimation for an individual. Population means reflect these multiple variables, many of which are interactive. These multiple variables would have the greatest effect on correlations derived from cross-cultural sampling, as in this study. As shown in our previous analysis, however, the correlations derived from within group data (ages 1–5 and 6–18 in each of the two inner-city

areas) were statistically comparable, although they predicted a greater change in PbB as PbA increased.

Since environmental quality is measured by community sampling, not individual environmental sampling, dust fall and surface dust lead are often the only composite index of the many variables that affect ingested lead. The environmental values assigned to 6- 18-year-old subjects at Omaha sites C (commercial) and S (suburban) in Table 1 suggest that the mean air lead of 0.9 $\mu g/m^3$ in area C vs. 0.3 $\mu g/m^3$ in area S is associated with an additional soil lead exposure of 422 $\mu g/g$ (519 − 97 = 422) and 300 $\mu g/g$ of extra house dust lead exposure (624 − 324 = 300). Substitution of these numbers in the linear description of the data for ages 1–18 years,

$$\ln PbB = \ln (15.7 + 1.92\ PbA + 0.00681\ PbS + 0.00718\ PbHD),$$

predicts, from the excess exposure of area C:

$$\begin{aligned} \ln PbB &= \ln (15.7 + 1.92\ (1) + 0.00681\ (422) + 0.00718\ (300)) \\ &= \ln (15.7 + 1.92 + 2.87 + 2.15) \\ &= \ln (15.7 + 1.92 + 5.02) \\ &= \ln (22.64). \end{aligned}$$

This response in PbB to an increase of 1.0 $\mu g/m^3$ PbA is 5.02 $\mu g/dl$ more than the predicted response of 1.92 $\mu g/dl$ to a 1-$\mu g/m^3$ increase in air lead with no increase in PbS and PbHD. This hypothetical blood:air slope of 6.94 (5.02 + 1.92) is relatively close to the blood:air slope of 4.40 predicted by the linear equation describing the data for ages 6–18, the group that includes all of the observations for site C:

$$\ln PbB = \ln (16.2 + 4.4\ PbA + 0.00457\ PbS + 0.00336\ PbHD).$$

The prediction of increases in total exposure with increases in air lead is consistent with the studies reported by Yankel *et al.* (1977) for children in the Idaho smelter area and by Roels *et al.* (1980) for school children in Belgium even though different variables modified the response. In the Idaho study, clear evidence was found for increased lead exposure in dustier households. Since general household dustiness as well as the lead content of soil and dust was highest in the immediate proximity of the smelter, there was a dual effect on lead exposure. In the Belgian study, socio-economic and housing variables were considered minimal, yet air lead correlated with soil lead and both were directly related to hand dust lead, the environmental variable that correlated most strongly with blood lead.

The large scale studies in Turin by Faschetti *et al.* (1983) of the effects of a change in the isotopic ratio of the lead added to gasoline similarly delineate the impact of air lead on the total daily burden of ingested lead. The Turin study provides strong evidence that air lead has a rapid and measurable effect on ingested lead, even in adult populations at low levels of air lead. It is consistent with the consensus that community studies describe steeper blood:air slopes than inhalation studies. Models of total exposure that relate blood lead to both inhaled and ingested lead would seem to provide a better prediction of the effect of changes in air quality.

Given the many individual and environmental variables that determine the contribution of fallout lead to surface lead as well as the approximation inherent in single site, averaged environmental data, a correlation that accounts for only 20 to 26% of the variability in childhood blood lead can still provide useful information. A linear model to describe the response of individual blood leads to averaged community exposure has the advantage of simplicity and avoids the mathematical limitations of the power model for calculations at zero levels of exposure. The equally good fit of the linear and power models for the Omaha data is consistent with Snee's prediction of the validity of this analysis (Snee, 1982a).

The linear model of total exposure employed in this analysis differs slightly from that of Snee (1982a), who proposed:

$$\mathrm{PbB} = A\,(\mathrm{PbAir} + \beta_1\,\mathrm{PbFood} + \beta_2\,\mathrm{PbWater} + \ldots.)^k.$$

In the absence of data on lead exposure from other sources, a single coefficient B is employed,

$$B = (\beta_1\,\mathrm{PbFood} + \beta_2\,\mathrm{PbWater} + \ldots).$$

The resulting model is

$$\mathrm{PbB} = A\,(\mathrm{PbAir} + B)^k.$$

The use of a common exponent k suggest an equivalent absorption of inhaled and ingested lead. The linear total exposure model of this study was modified to be biologically more appropriate to the greater absorption of inhaled lead and to permit estimation of the relative contribution of various sources of ingested lead.

CONCLUSION

Investigation of a linear total exposure model to describe the response of blood lead to environmental lead in the Omaha Study of children ages 1–18 years demonstrates a data fit that is comparable to the power function model. This linear model, $\ln \mathrm{PbB} = \ln(\beta_0 + \beta_1\,\mathrm{PbAir} + \beta_2\,\mathrm{PbSoil} + \beta_3\,\mathrm{PbHouse\ Dust})$, as applied to the Omaha data, predicts that blood lead increases 1.92 μg/dl as air lead increases 1.0 $\mu g/m^3$ if other levels of exposure are constant. Since prior and current atmospheric fallout directly modify the daily burden of ingested lead, larger changes in blood lead are predicted by the associated changes in the surface deposition of lead. The linear model avoids the mathematical limitations of the power model which predicts a blood lead of zero at an air lead of zero. The linear model of total exposure deserves investigation in other community studies as an environmentally valid description of the effect on blood lead of the obligatory intercorrelation of air lead and atmospheric fall out. If it can be applied with equal confidence to similarly representative data, the blood:air dose response predicted by the linear model can provide a rational basis for an air quality standard for lead.

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